09/544,045
AU#18

WEST Search History

DATE: Saturday, August 31, 2002

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ		
L10	l7 or L9	55	L10
L9	mutant near5 resolvase	10	L9
L8	mutant resolvase	4	L8
L7	mutant near5 recombinase	49	L7
L6	l4 with L5	93	L6
L5	l2 near3 L1	330	L5
L4	l2 near5 L3	20515	L4
L3	site	443835	L3
L2	mutant or mutat$ or deletion or insertion or substitution or loxp	812398	L2
L1	recombinase or resolvase or integrase or telomerase	3295	L1

END OF SEARCH HISTORY

09/544045
Amdt B

=> s recombinase? or integrase? or telomerase? or resolvase?
L1 30130 RECOMBINASE? OR INTEGRASE? OR TELOMERASE? OR RESOLVASE?

=> s mutat? or mutant? or loxP
L2 1336280 MUTAT? OR MUTANT? OR LOXP

=> s l1 and l2
L3 7361 L1 AND L2

=> s l1(3n)l2
L4 1473 L1(3N) L2

=> s site?
L5 2502261 SITE?

=> s l5(3n)l2
L6 57826 L5(3N) L2

=> s l4 and l6
L7 310 L4 AND L6

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8 178 DUP REM L7 (132 DUPLICATES REMOVED)

=> s l8 and py<1999
1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L9 85 L8 AND PY<1999

=> d l9 ibib abs 1-85

L9 ANSWER 1 OF 85 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:59332 BIOSIS
DOCUMENT NUMBER: PREV199900059332
TITLE: Non-autonomy of AGAMOUS function in flower development: Use of a Cre/loxP method for mosaic analysis in Arabidopsis.
AUTHOR(S): Sieburth, Leslie E. (1); Drews, Gary N.; Meyerowitz, Elliot M.
CORPORATE SOURCE: (1) Dep. Biol., McGill Univ., 1205 Dr. Penfield Ave., Montreal, PQ H3A 1B1 Canada
SOURCE: Development (Cambridge), (***Nov., 1998***) Vol. 125, No. 21, pp. 4303-4312.
ISSN: 0950-1991.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Angiosperms use a multi-layered meristem (typically L1, L2 and L3) to produce primordia that then develop into plant organs. A number of experiments show that communication between the cell layers is important for normal development. We examined whether the function of the flower developmental control gene AGAMOUS involves communication across these layers. We developed a mosaic strategy using the Cre/ ***loxP*** ***site*** -specific ***recombinase*** system, and identified the sector structure for mosaics that produced mutant flowers. The major conclusions were that (1) AGAMOUS must be active in the L2 for staminoid and carpelloid tissues, (2) that AGAMOUS must be active in the L2 and the L3 for floral meristem determinacy, and (3) that epidermal cell identity can be communicated by the L2 to the L1 layer.

L9 ANSWER 2 OF 85 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:28032 BIOSIS
DOCUMENT NUMBER: PREV199900028032
TITLE: Structural basis for inactivating mutations and pH-dependent activity of avian sarcoma virus integrase.
AUTHOR(S): Lubkowski, Jacek; Yang, Fan; Alexandratos, Jerry; Merkel, George; Katz, Richard A.; Gravuer, Kelly; Skalka, Anna Marie; Wlodawer, Alexander (1)
CORPORATE SOURCE: (1) Macromolecular Structure Lab., ABL Basic Res. Program, NCI-Frederick Cancer Res. Development Center, National Inst. Health, Frederick, MD 21702 USA
SOURCE: Journal of Biological Chemistry, (***Dec. 4, 1998***) Vol. 273, No. 49, pp. 32685-32689.
ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Crystallographic studies of the catalytic core domain of avian sarcoma virus integrase (ASV IN) have provided the most detailed picture so far of the active site of this enzyme, which belongs to an important class of targets for designing drugs against AIDS. Recently, crystals of an inactive D64N mutant were obtained under conditions identical to those used for the native enzyme. Data were collected at different pH values and in the presence of divalent cations. Data were also collected at low pH for the crystals of the native ASV IN core domain. In the structures of native ASV IN at pH 6.0 and below, as well as in all structures of the D64N mutants, the side chain of the active site residue Asx-64 (Asx denotes Asn or Asp) is rotated by apprx 150degree around the Calpha-Cbeta bond, compared with the structures at higher pH. In the new structures, this residue makes hydrogen bonds with the amide group of Asn-160, and thus, the usual metal-binding site, consisting of Asp-64, Asp-121, and Glu-157, is disrupted. Surprisingly, however, a single Zn2+ can still bind to Asp-121 in the mutant, without restoration of the activity of the enzyme. These structures have elucidated an unexpected mechanism of inactivation of the enzyme by lowering the pH or by mutation, in which a protonated side chain of Asx-64 changes its orientation and interaction partner.

L9 ANSWER 3 OF 85 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:269965 BIOSIS
DOCUMENT NUMBER: PREV199800269965
TITLE: Effects of mutations in residues near the active site of human immunodeficiency virus type 1 integrase on specific enzyme-substrate interactions.
AUTHOR(S): Gerton, Jennifer L.; Ohgi, Sharron; Olsen, Mari; Derisi, Joseph; Brown, Patrick O. (1)
CORPORATE SOURCE: (1) B253 Beckman Center, Stanford Univ. Med. Center, Stanford, CA 94305-5428 USA
SOURCE: Journal of Virology, (***June, 1998***) Vol. 72, No. 6, pp. 5046-5055.
ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The phylogenetically conserved catalytic core domain of human immunodeficiency virus type 1 (HIV-1) integrase contains elements necessary for specific recognition of viral and target DNA features. In order to identify specific amino acids that determine substrate specificity, we mutagenized phylogenetically conserved residues that were located in close proximity to the active-site residues in the crystal structure of the isolated catalytic core domain of HIV-1 integrase. Residues composing the phylogenetically conserved DD(35)E active-site motif were also mutagenized. Purified mutant proteins were evaluated for their ability to recognize the phylogenetically conserved CA/TG base pairs near the viral DNA ends and the unpaired dinucleotide at the 5' end of the viral DNA, using disintegration substrates. Our findings suggest that specificity for the conserved A/T base pair depends on the active-site residue E152. The phenotype of IN(Q148L) suggested that Q148 may be involved in interactions with the 5' dinucleotide of the viral DNA end. The activities of some o t proteins with mutations in residues in close proximity to the active-site aspartic and glutamic acids were salt Sensitive, suggesting that these mutations disrupted interactions with DNA.

L9 ANSWER 4 OF 85 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:76893 BIOSIS
DOCUMENT NUMBER: PREV199800076893
TITLE: Spatio-temporally controlled site-specific somatic mutagenesis in the mouse.
AUTHOR(S): Brocard, Jacques; Warot, Xavier; Wendling, Olivia;

=> s recombinase? or transposase?
L1 13583 RECOMBINASE? OR TRANSPOSASE?

=> s variant or mutant or mutat? or modify or modified
L2 2380880 VARIANT OR MUTANT OR MUTAT? OR MODIFY OR MODIFIED

=> s l1(l)l2
L3 3331 L1(L) L2

=> s mutant recombinase?
L4 3 MUTANT RECOMBINASE?

=> s mutant(3n)recombinase?
L5 61 MUTANT(3N) RECOMBINASE?

=> s variant(3n)recombinase?
L6 8 VARIANT(3N) RECOMBINASE?

=> s mutant(3n)transposase?
L7 124 MUTANT(3N) TRANSPOSASE?

=> s variant(3n)transposase?
L8 19 VARIANT(3N) TRANSPOSASE?

=> s l5 or l6 or l7 or l8
4 FILES SEARCHED...
L9 210 L5 OR L6 OR L7 OR L8

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 88 DUP REM L9 (122 DUPLICATES REMOVED)

=> s l10 and py<1999
1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L11 65 L10 AND PY<1999

=> d l11 ibib abs 1-65

L11 ANSWER 1 OF 65 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:473013 BIOSIS
DOCUMENT NUMBER: PREV199800473013
TITLE: Cre mutants with altered DNA binding properties.
AUTHOR(S): Hartung, Markus; Kisters-Woike, Brigitte (1)
CORPORATE SOURCE: (1) Inst. Genetics, Univ. Cologne, Weyertal 121, D-50931
Cologne Germany
SOURCE: Journal of Biological Chemistry, (***Sept. 4, 1998***) Vol. 273, No. 36, pp. 22884-22891.
ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The recombinase Cre of bacteriophage PI is a member of the family of site-specific recombinases and integrases that catalyze inter- and intramolecular DNA rearrangements. To understand how this protein specifically recognizes its target sequence, we constructed Cre mutants with amino acid substitutions in different positions of the presumptive DNA binding region. Here we present the results of in vitro DNA binding and in vivo recombination experiments with these Cre mutants. Most substitutions of presumptive DNA-binding amino acids in in vitro tests resulted either in the loss of target binding or in a broadening of target recognition specificity. Of the mutations resulting in a broadening of target specificity, one, N317A, results in a reduced recombination efficiency with the wild-type loxP target but recombines, in contrast to wild-type Cre, in in vivo experiments, with a symmetric variant of the wild-type target sequence. This target variant differs from wild-type loxP by the symmetric C to A replacement in position 6 of the inverted repeats. We propose a common multihelical DNA binding motif for the family of integrases and recombinases. This model implies a major structural rearrangement for the DNA binding region of lambda integrase, analogous to the structural rearrangements of the DNA binding motifs of other proteins when contacting their target DNA.

L11 ANSWER 2 OF 65 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:448150 BIOSIS
DOCUMENT NUMBER: PREV199800448150
TITLE: Mutations in domain IIIalpha of the Mu transposase: Evidence suggesting an active site component which interacts with Mu-host junction.
AUTHOR(S): Naigamwalla, Darius Z.; Coros, Colin J.; Wu, Zhenguo; Chaconas, George (1)
CORPORATE SOURCE: (1) Dep. Biochem., Univ. Western Ontario, London, ON N6A
5C1 Canada
SOURCE: Journal of Molecular Biology, (***Sept. 18, 1998***) Vol. 282, No. 2, pp. 265-274.
ISSN: 0022-2836.
DOCUMENT TYPE: Article
LANGUAGE: English
AB A series of point mutations was constructed in domain IIIalpha of the Mu A protein. The ***mutant*** ***transposases*** were purified and assayed for their ability to promote various aspects of the in vitro Mu DNA strand transfer, reaction. All mutants with discernable phenotypes were inhibited in stable synapsis (Type 0 or Type 1 complex formation). In contrast, these mutant proteins were capable of LER formation (a transient early reaction intermediate in which the Mu left and right ends have been synapsed with the enhancer), at levels comparable to wild-type transposase. These proteins therefore comprise a novel class of transposase mutants, which are specifically inhibited in stable transpososome assembly. The defect in these proteins was also uniformly suppressed by either Mn2+, or the Mu B protein in the presence of ATP and target DNA. Striking phenotypic similarities were recognized between the domain IIIalpha ***transposase*** ***mutant*** characteristics noted above, and those for substrate mutants carrying a terminal base-pair substitution at the point of cleavage on the donor molecule. This phenotypic congruence suggests that the alterations in either protein or DNA are exerting an effect on the same step of the reaction i.e., engagement of the terminal nucleotide by the active site. We suggest that domain IIIalpha of the transposase comprises the substrate binding pocket of the active site which interacts with the Mu-host junction.

L11 ANSWER 3 OF 65 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:390367 BIOSIS
DOCUMENT NUMBER: PREV199800390367
TITLE: Altering the DNA-binding specificity of Mu transposase in vitro.
AUTHOR(S): Namgoong, Soon-Young; Sankaralingam, Senthil; Harshey,
Rasika M. (1)
CORPORATE SOURCE: (1) Dep. Microbiol., Univ. Texas Austin, Austin, TX 78712
USA
SOURCE: Nucleic Acids Research, (***Aug. 1, 1998***) Vol. 26, No. 15, pp. 3521-3527.
ISSN: 0305-1048.
DOCUMENT TYPE: Article
LANGUAGE: English
AB We describe the isolation of a ***variant*** of Mu ***transposase*** (MuA protein) which can recognize altered att sites at the ends of Mu DNA.
No prior knowledge of the structure of the DNA binding domain or its mode of interaction with att DNA was necessary to obtain this variant. Protein secondary structure programs initially helped target mutations to predicted helical regions within a subdomain of MuA demonstrated to harbor att DNA binding activity. Of the 54 mutant positions examined, only two showed decreased affinity for att DNA, while eight others affected assembly of the Mu transpososome. A variant impaired in DNA binding (MuA(R146V)), and predicted to be in the recognition helix of an HTH motif, was challenged with altered att sites created from degenerate oligonucleotides to select for novel DNA binding specificity. DNA sequences bound to MuA(R146V) were detected by gel-retardation, and following several steps of PCR amplification/enrichment, were identified by cloning and sequencing. The strategy allowed recovery of an altered att

09/544045

=> s recombinase
L1 9110 RECOMBINASE

=> s integrase
L2 8270 INTEGRASE

=> s l1 or l2
L3 16036 L1 OR L2

=> s mutant? or mutat? or variant?
L4 1692740 MUTANT? OR MUTAT? OR VARIANT?

=> s l3(5n)l4
L5 924 L3(5N) L4

=> s decreas? or reduc? or limit? or inefficient?
3 FILES SEARCHED...
L6 11920258 DECREAS? OR REDUC? OR LIMIT? OR INEFFICIENT?

=> s l5 and l6

L7 161 L5 AND L6

=> dup rem l7

PROCESSING COMPLETED FOR L7
L8 75 DUP REM L7 (86 DUPLICATES REMOVED)

=> s l8 and py<2000
1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L9 43 L8 AND PY<2000

=> d l9 ibib abs 1-43

L9 ANSWER 1 OF 43 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:57016 BIOSIS
DOCUMENT NUMBER: PREV199900057016
TITLE: ***Mutations*** in nonconserved domains of Ty3 ***integrase*** affect multiple stages of the Ty3 life cycle.
AUTHOR(S): Nymark-Mcmahon, M. Henrietta; Sandmeyer, Suzanne B. (1)
CORPORATE SOURCE: (1) Dep. Biol. Chem., Univ. California Irvine, 240D Med. Sci. I, Irvine, CA 92697-1700 USA
SOURCE: Journal of Virology, (***Jan., 1999***) Vol. 73, No. 1, pp. 453-465.
ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Ty3, a retroviruslike element of Saccharomyces cerevisiae, transposes into positions immediately upstream of RNA polymerase III-transcribed genes. The Ty3 integrase (IN) protein is required for integration of the replicated, extrachromosomal Ty3 DNA. In retroviral IN, a conserved core region is sufficient for strand transfer activity. In this study, charged-to-alanine scanning mutagenesis was used to investigate the roles of the nonconserved amino- and carboxyl-terminal regions of Ty3 IN. Each of the 20 IN mutants was defective for transposition, but no mutant was grossly defective for capsid maturation. All mutations affecting steady-state levels of mature IN protein resulted in ***reduced*** levels of replicated DNA, even when polymerase activity was not grossly defective as measured by exogenous reverse transcriptase activity assay. Thus, IN could contribute to nonpolymerase functions required for DNA production in vivo or to the stability of the DNA product. Several mutations in the carboxyl-terminal domain resulted in relatively low levels of processed 3' ends of the replicated DNA, suggesting that this domain may be important for binding of IN to the long terminal repeat. Another class of mutants produced wild-type amounts of DNA with correctly processed 3' ends. This class could include mutants affected in nuclear entry and target association. Collectively, these mutations demonstrate that in vivo, within the preintegration complex, IN performs a central role in coordinating multiple late stages of the retrotransposition life cycle.

L9 ANSWER 2 OF 43 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:17963 BIOSIS
DOCUMENT NUMBER: PREV199900017963
TITLE: The frequency of illegitimate V(D)J ***recombinase*** -mediated ***mutations*** in children treated with etoposide-containing antileukemic therapy.
AUTHOR(S): Fuscoe, James C. (1); Knapp, Geremy W.; Hanley, Nancy M.; Setzer, R. Woodrow; Sandlund, John T.; Pui, Ching-Hon; Relling, Mary V.
CORPORATE SOURCE: (1) Environ. Carcinogenesis Div., Mail Drop 68, Natl. Health Environ. Effects Res. Lab., U.S. Environ. Protection Agency, Research Triangle Park, NC 27711 USA
SOURCE: Mutation Research, (***Nov. 9, 1998***) Vol. 419, No. 1-3, pp. 107-121.
ISSN: 0027-5107.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Etoposide is among the most widely used anti-cancer drugs. Its use, however, has been associated with increased risk of secondary acute myeloid leukemia (AML) which is characterized by chromosomal translocations suggesting involvement of recombination-associated motifs at the breakpoints. A PCR-based assay was developed to quantitate the frequency of two illegitimate V(D)J recombinase-mediated genomic rearrangements-a 20-kb deletion in the hprt gene and the bcl2/IgH translocation (t(14;18)) found in non-Hodgkin's lymphoma. We examined both lymphocyte and non-lymphocyte blood cell DNA of children with acute lymphoblastic leukemia (ALL) for changes in the frequencies of these biomarkers during etoposide therapy to determine the level of illegitimate V(D)J recombination changes during therapy. A low level of t(14;18) was found in the lymphocytes before etoposide treatment, which was significantly ***reduced*** during etoposide therapy, In before-etoposide samples, no t(14;18) were found among 7.72 X 107 non-lymphocytes; during treatment none were found among 1.87 X 108 non-lymphocytes. Deletions were not found before etoposide treatment in either the lymphocytes (6.67 X 107) or non-lymphocytes (5.43 X 107) and were non-significantly elevated during etoposide therapy (1 in 1.4 X 108 lymphocytes and 1 in 1.39 X 108 non-lymphocytes). It is interesting to note the one patient with an hprt deletion mutation in non-lymphocytes; V(D)J recombination is not normally found in this cell type, but is the cell type from which AML derives. Several patients had clones of t(14;18)-bearing cells as determined by DNA sequence analysis. These results suggest that this etoposide-based chemotherapy was ineffective in producing genomic rearrangements mediated by illegitimate V(D)J recombination in these patients.

L9 ANSWER 3 OF 43 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:473013 BIOSIS
DOCUMENT NUMBER: PREV199800473013
TITLE: Cre mutants with altered DNA binding properties.
AUTHOR(S): Hartung, Markus; Kisters-Woike, Brigitte (1)
CORPORATE SOURCE: (1) Inst. Genetics, Univ. Cologne, Weyertal 121, D-50931 Cologne Germany
SOURCE: Journal of Biological Chemistry, (***Sept. 4, 1998***) Vol. 273, No. 36, pp. 22884-22891.
ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The recombinase Cre of bacteriophage PI is a member of the family of site-specific recombinases and integrases that catalyze inter- and intramolecular DNA rearrangements. To understand how this protein specifically recognizes its target sequence, we constructed Cre mutants with amino acid substitutions in different positions of the presumptive DNA binding region. Here we present the results of in vitro DNA binding and in vivo recombination experiments with these Cre mutants. Most substitutions of presumptive DNA-binding amino acids in in vitro tests resulted either in the loss of target binding or in a broadening of target recognition specificity. Of the mutations resulting in a broadening of